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Case report

Coronary blood flow and perfusion pressure during coronary angiography in patients with ongoing mechanical chest compression: A report on 6 cases☆

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ABSTRACT

Patients with pulseless electrical activity or refractory ventricular fibrillation have a very bad prognosis. Coronary angiography and angioplasty may be required to restore an effective circulation, but this must be performed whilst chest compressions are continued. The LUCAS chest compression device is suitable for this purpose. So far there are no reports on the effect of this device on coronary circulation in humans. We monitored the coronary perfusion pressure assessed invasively as the difference between the diastolic pressures at the coronary ostium and right atrium, and compared these pressures with coronary flow graded using the TIMI scale in 6 patients. In 4 out of 6 we found a satisfactory coronary artery perfusion pressure and TIMI grade 3 flow (normal) on coronary angiography. Two of these patients survived the first 24 h. Two patients did not have a satisfactory perfusion pressure and adequate flow rate was not seen.

1. Introduction

Mechanical chest compression may be necessary to make percutaneous coronary intervention (PCI) possible during ongoing resuscitation. The LUCAS device (LUCAS 1, Jolife, Lund, Sweden), is a gas-driven sternal compression–decompression machine.[1] As the LUCAS device is relatively translucent for x-radiation it is well suited for use during PCI,[2] and it is capable of producing an adequate systemic blood pressure without excessive injuries.[3] However, the ultimate goal for the device in addition to ensuring an adequate cerebral circulation[4] is to generate a satisfactory coronary perfusion pressure to achieve a sufficient coronary blood flow; ideally angiographically documented TIMI (thrombolysis in myocardial infarction) Grade 3 flow that represents normal flow and run-off. The subsequent improved cardiac aerobic metabolism during and after coronary angiography and PCI may in turn increase the possibility for return of spontaneous circulation (ROSC).[5]

An adequate coronary perfusion pressure is dependent on a difference between the diastolic blood pressure in the coronary artery and the coronary sinus. This may be monitored using a catheter engaged in the coronary ostium and a catheter in the right atrium at the level of the coronary sinus. The generated coronary blood flow may be verified with coronary angiography using the TIMI flow grading. To evaluate if we managed to perfuse the heart during ongoing mechanical chest compression to extend an otherwise hopeless resuscitation, the relationship between angiographically documented TIMI flow and invasively obtained measures of coronary perfusion pressure was assessed in 6 patients.

2. Patients

2.1. Patient 1

Patient 1 (57-year-old diabetic male) who was admitted with ST-elevation myocardial infarction (STEMI) lost consciousness due

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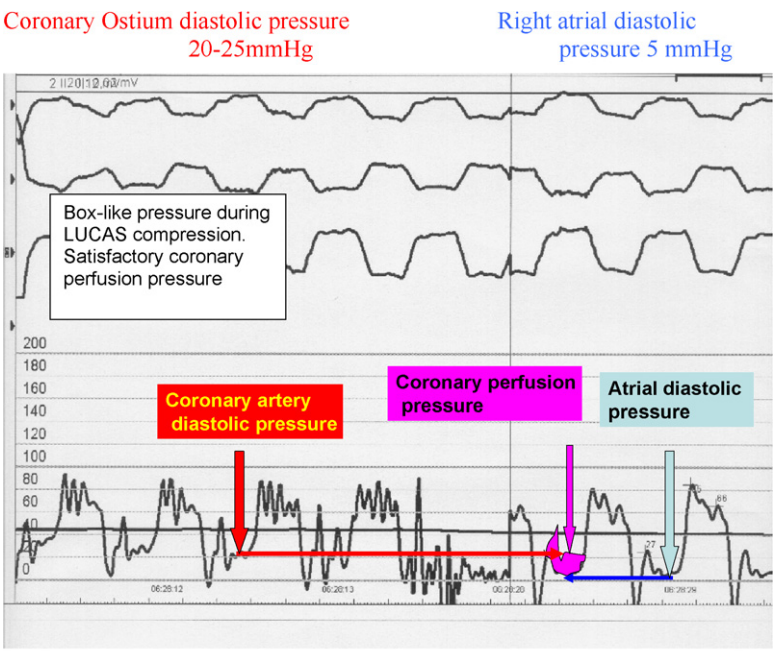

Fig. 1. Effect of LUCAS during asystole in pat. No. 1 showing difference between diastolic blood pressure in the coronary ostium and the right atrium, generating a coronary perfusion pressure of approximately 15–20 mmHg.

to ventricular fibrillation (VF) immediately after the diagnosis was verified. Successful defibrillation was performed and ROSC was promptly obtained. As VF recurred necessitating several defibrillations despite a total dosage of 450 mg of amiodarone, LUCAS was applied to ensure adequate compressions during transport to the catheterization laboratory. Despite an adequate systolic pressure obtained with the LUCAS device at arrival, electrical asystole was diagnosed. Angiography revealed no occlusions, but showed a 70% stenosis in the left anterior descending artery (LAD) associated with TIMI 3 flow during mechanical chest compression. Therefore, PCI was not performed. Mechanical chest compression with the LUCAS device was continued and tribonate (buffer) was administered to treat severe acidosis; pH 6.55. Fig. 1 demonstrates the pressures in the right atrium and the proximal part of the left coronary artery from which a coronary perfusion pressure gradient of approximately 27 mmHg can be calculated. After approximately 30 min of LUCAS compression the patient developed a slow heart rhythm with broad complexes, but following the injection of adrenalin 1 mg and atropine 1 mg sinus rhythm was achieved and he was haemodynamically stabilized. The patient was cooled for the first 24 h and medically treated in accordance with the new guidelines.[6] He regained consciousness on day 2 and answered simple questions adequately. Unfortunately he sustained a further asystolic arrest and died on the third day. Autopsy showed no extensive intrathoracic or intra-abdominal injuries, but a large sub-endocardial infarction was diagnosed.

2.2. Patient 2

Patient 2 (48-year-old healthy male) was admitted in VF. Cardiopulmonary resuscitation (CPR) was immediately initiated and direct current (DC) shock had to be given several times due to recurrent VF. During mechanical chest compression with the LUCAS device coronary angiography revealed normal coronary arteries with TIMI 3 flow in both the right and the left coronary artery. Fig. 2 reveals that the dye is reaching the end segments of the arteries within a few cardiac cycles during chest compression with the LUCAS device. The coronary perfusion pressure was calculated to be approximately 25 mmHg, a pressure gradient sufficient for defibrillation to occur. After successful defibrillation the patient was haemodynamically stabilized. Intra-thoracic bleeding caused by a mechanically induced tear of the mammary artery was surgically treated. A haematogram revealed thrombocytosis. Unfortunately, the patient died 48 h following hospitalization due to ileus. His primary condition was diagnosed as post-infectious phagocytic macrophage syndrome.

2.3. Patient 3

Patient 3 (80-year-old male) was admitted with STEMI complicated by cardiogenic shock. Intra-aortic balloon pump (IABP) was inserted and the LUCAS device was applied due to a systolic blood pressure of 40 mmHg and loss of consciousness. Angiography revealed severe triple vessel disease; chronically occluded right coronary artery, subtotal occlusion of the left anterior descending artery and thrombotic occlusion of the circumflex artery. Percutaneous coronary intervention (PCI) was performed with ongoing LUCAS chest compression, ventilation and IABP counter pulsation, but we only achieved TIMI 2 flow in the infarct related vessel and the calculated coronary perfusion pressure was 5 mmHg. This patient had extensive coronary artery disease and was never haemodynamically stabilized. Patients 2 and 3 have previously been reported.[3]

2.4. Patient 4

Patient 4 (51-year-old previously healthy male) experienced sudden cardiac arrest at home. Resuscitation was immediately